# In the United States Court of Federal Claims

**OFFICE OF SPECIAL MASTERS**

No. 15-1016V
Filed: December 12, 2017
Not to be Published

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| MARYELLEN KOTTENSTETTE and NICHOLAS KOTTENSTETTE as best friends of their daughter (CK), | * | |
| Petitioners, | * | |
| v. | * | Diphtheria-tetanus-acellular pertussis ("DTaP"), haemophilus B influenzae ("HiB"), inactivated polio vaccine ("IPV"), and pneumococcal vaccine ("Prevnar"); cryptogenic infantile spasms |
| SECRETARY OF HEALTH AND HUMAN SERVICES, | * | |
| Respondent. | * | |

John F. McHugh, New York, NY, for petitioners.
Camille M. Collett, Washington, DC, for respondent.

**MILLMAN, Special Master**

## RULING ON ENTITLEMENT[1]

On September 11, 2015, petitioners filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012), alleging that diphtheria-tetanus-acellular pertussis ("DTaP"), haemophilus B influenza ("HiB"), inactivated polio vaccine ("IPV"), and pneumococcal ("Prevnar") vaccines administered to their daughter CK on October 2, 2012, caused her a Table encephalopathy or, in the alternative, a non-Table encephalopathy, and

[1] Because this unpublished decision contains a reasoned explanation for the special master's action in this case, the special master intends to post this unpublished decision on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). Vaccine Rule 18(b) states that all decisions of the special masters will be made available to the public unless they contain trade secrets or commercial or financial information that is privileged and confidential, or medical or similar information whose disclosure would constitute a clearly unwarranted invasion of privacy. When such a decision is filed, petitioner has 14 days to identify and move to redact such information prior to the document's disclosure. If the special master, upon review, agrees that the identified material fits within the banned categories listed above, the special master shall redact such material from public access.

infantile spasms. Pet. at ¶¶ 4, 5-7, 10.

On November 20, 2015, the undersigned held the first telephonic status conference in this case and encouraged the parties to settle. The undersigned gave petitioner until April 15, 2015 to make a demand on respondent.

On August 10, 2016, petitioners filed a CD containing the expert report of Dr. Marcel Kinsbourne, with attached medical articles. A day later, after petitioners moved for numerous extensions of time to make a demand, they made a demand on respondent on August 11, 2016.

Although each party had prepared life care plans, respondent wanted to file a Rule 4(c) Report and an expert report. The undersigned gave respondent until November 7, 2016 to file a Rule 4(c) Report and an expert report.

Respondent moved for numerous extensions of time to file a Rule 4(c) Report and an expert report and, on February 6, 2017, filed his Rule 4(c) Report and the expert report of Dr. John Zempel, with attached medical articles. On February 14, 2017, during a telephonic status conference, respondent's counsel stated HHS was not interested in settlement.

On April 10, 2017, petitioners filed a supplemental expert report from Dr. Kinsbourne.

On July 31, 2017, the undersigned held a hearing in this case. Testifying for petitioners were Mrs. Kottenstette and Dr. Marcel Kinsbourne, a pediatric neurologist. Testifying for respondent was Dr. John Zempel, a pediatric neurologist and pediatric epileptologist.

On September 12, 2017, petitioners filed a post-hearing brief.

On November 27, 2017, respondent filed a post-hearing brief.

The undersigned finds that petitioners have prevailed on their allegations that CK's vaccinations administered on October 2, 2012 caused her afebrile infantile spasms and a non-Table chronic encephalopathy. Petitioners have not prevailed on their allegation that CK had a Table encephalopathy.

## FACTS

On June 1, 2012, CK was born.

On October 2, 2012, at the age of four months, CK received DTaP, HiB, IPV, and Prevnar vaccinations. Med. recs. Ex. 2, at 21.

Later on October 2, 2012, CK was taken to the University of Massachusetts Children's Medical Center because she was having abnormal arm and shoulder movements multiple times

that evening. Med. recs. Ex. 4, at 1. Her temperature was 98.4 degrees. Id. At 8:30 p.m., she had repetitive jerking arm movements for about five minutes with bilateral shrugging and then a hugging motion. Id. at 9. CK was alert. Her eyes or legs were not involved in these movements. She did not have a post-ictal state. She had no change in urinary output or bowel movement. She did not have fevers, chills, or fussiness. She had received DPT, HiB, polio, and pneumonia vaccinations that day. She was sleeping quietly. CK was alert and oriented. She looked well. Id. Her family history was an uncle with epilepsy. Id. at 10. CK was neurologically and developmentally normal. She had a normal well-child check-up that day. She had normal tone and 2+ reflexes. She had been previously well. She had been behind on her immunizations, receiving her two-month vaccinations that day. She was alert and active. The diagnosis was rhythmic movement/possible seizure. Id.

From October 6-10, 2012, CK was at Boston Children's Hospital. Med. recs. Ex. 2, at 1.[2] Dr. Irina M. Anselm wrote the discharge summary. CK had received vaccinations on Tuesday morning and was mildly fussy, but otherwise well. She did not have fever or signs of illness. She awoke out of sleep and suddenly had a series of jerks with her arms extended outward and jerking inward every five seconds for about three to five minutes. She seemed to be alert throughout the entire episode. Afterward she went back to baseline immediately. Her pediatrician recommended against an EEG because the pediatrician felt that the episode was just a mild reaction to the vaccinations. CK had a second episode that evening that was again three to five minutes long, but the mother did not seek further medical attention due to her discussion with the pediatrician. On October 6, 2012, at around 5:30 a.m., CK had another three- to five-minute episode which was captured on video. Dr. Anselm watched the video. CK was in her mother's arms, looking around, and was appropriately alert, with intermittent episodes of rapid arm extension and then shoulder abduction and arm jerks inwards. These movements were consistent with infantile spasms occurring every 10-15 seconds on the video. CK had otherwise been well appearing and in her usual state of health. She was otherwise a healthy baby who had been feeding and growing well and progressing appropriately. Id. An EEG result was consistent with hypsarrhythmia. Id. at 3. She was prescribed ACTH and her parents were instructed that CK was not to have immunizations for six months. Id.

On October 30, 2012, CK saw Dr. Michel N. Fayad, a neurologist. Id. at 10. The history was CK was alert and well during her first episode of infantile spasms. She did not have any regression in development. Id. at 11. She was very alert, smiled, laughed, vocalized, and reached for objects frequently. She had a history of reflux. She has a maternal uncle with difficult-to-control seizures which CK's mother believed a lesion caused. CK had a paternal second cousin's daughter with seizures since she was young. On physical examination, CK was extremely alert and made excellent eye contact with her parents and Dr. Fayad. Id. On physical examination, CK had mildly increased tone in both legs. Id. at 12. The EEG result showed an abnormal background but did not meet the criteria for hypsarrhythmia. However, her

[2] Petitioners' counsel filed this collection of records as Exhibit 3, but marked each page as Exhibit 2. This would be the second Exhibit 2 since the first Exhibit 2 consists of CH's pediatric records. For ease of reference to the Boston Children's Hospital records, the undersigned refers to these pages as Exhibit 2 as petitioners' counsel marked them.

presentation was consistent with infantile spasms. Id.

As of June 22, 2017, CK has physical disabilities that impact her functional mobility, postural stability, eye-hand coordination, fine motor control, pre-writing skills, and self-care skills. Med. recs. Ex. 16, at 5. CK also has a visual impairment that affects her performance on visually-based activities. Id. CK can differentiate sounds and turn her head toward unfamiliar sounds, but she does not yet respond to her name. Id. at 9. She does not yet understand any words and does not yet use gestures to communicate. Id.

## EXPERT REPORTS AND MEDICAL LITERATURE

Petitioners filed an expert report from Dr. Marcel Kinsbourne, dated July 28, 2016. Ex. 6. He describes CK's refractory seizures as cryptogenic infantile spasms, meaning the cause is unknown. Relying on the Bellman study (1983),[3] based on the data underlying the National Children's Encephalopathy Study ("NCES"),[4] Dr. Kinsbourne states that DPT vaccine can trigger infantile spasms and thus accelerate the onset of infantile spasms in cryptogenic cases. Ex. 6, at 3.

The Bellman study is entitled Infantile Spasms and Pertussis Immunisation, LANCET 1:1031-34 (1983). M.H. Bellman, E.M. Ross, and D.L. Miller, the co-authors of this study, were also three of the five co-authors of the much larger NCES study and took data of the incidence of infantile spasms occurring post-DPT vaccination from the NCES data, dividing the infantile spasms children into groupings by one-week intervals for up to four weeks post-vaccination. They divided the cases as well by whether the infantile spasms were cryptogenic (unknown cause) or symptomatic (known cause). Id. at 1031. Whereas the NCES showed no significant association between DPT vaccination and onset of infantile spasms by looking at the entire 28-day period as a whole, Bellman and his co-authors in their separate study analyzed incidence of infantile spasms week by week. They found more cases of infantile spasms occurring within one week of vaccination compared to controls, whereas they also found fewer cases of infantile spasms occurring during the second week after vaccination compared to controls. The third and fourth weeks of onset of infantile spasms post-vaccination did not differ from the incidence among controls. Bellman and his co-authors surmised that pertussis vaccine "may precipitate the onset of spasms in those children in whom the disorder is already destined to develop." Id. at 1033. Bellman and his co-authors regarded vaccination as a trigger, rather than a cause, of infantile spasms. Id.

Dr. Kinsbourne also relies on the Melchior study (1977)[5] which analyzed the effect of a change in scheduling DPT vaccination in Denmark from initially 5 months of age to vaccination at 5 weeks of age. Ex. 6, at 3. When Danish children received DPT at age five months of age,

[3] Bellman is Ex. 6-4. Respondent filed the same article as Ex. D, Tab. 1.
[4] The National Childhood Encephalopathy Study. Whooping Cough, by R. Alderslade, M.H. Bellman, N.S.B. Rawson, E.M. Ross, and D.L. Miller (London: Her Majesty's Stationery Office, 1981).
[5] Melchior is Ex. 6-16. Respondent filed the same article as Ex. D, Tab 2.

the rate of infantile spasm onset before the age of two months was 12 percent. However, when the schedule changed and Danish children received DPT at five weeks of age, almost twice as many Danish children had onset of infantile spasms before the age of two months. Id.

The Melchior study is entitled Infantile spasms and early immunization against whooping cough. Danish survey from 1970 to 1975, 52 ARCH OF DIS IN CHILDHOOD 134-37 (1977). Melchior, noting the increase in the number of infantile spasms before the age of two months when the only variable was earlier vaccination, wrote that vaccination might have been a trigger mechanism in three cases of symptomatic infantile spasms. Id. at 135 (Table 2). He concludes "that a causal connection between whooping cough immunization and infantile spasms is very unlikely except in a few cases and that time-coincidence is the most likely factor . . . ." Id. at 136.

Citing Kivity,[6] Dr. Kinsbourne states that since CK had cryptogenic infantile spasms, her outcome without the DPT vaccine trigger would not have been necessarily poor, unlike those children with symptomatic infantile spasms in whom a poor outcome is expected. Ex. 6, at 3-4. Long-term Cognitive Outcomes of a Cohort of Children with Cryptogenic Infantile Spasms Treated with High-dose Adrenocorticotropic Hormone by S. Kivity, et al., 45 EPILEPSIA 3:255-62 (2004). Adrenocorticotropic hormone is also known as "ACTH."

Kivity and her co-authors compared children who had early treatment with ACTH of cryptogenic infantile spasms with the outcomes of those treated after one month of onset and found the former group had a favorable cognitive outcome. The authors focused solely on cryptogenic infantile spasms because children with symptomatic infantile spasms were more likely to have a poor intellectual outcome due to their underlying disorder. Id. at 255, 260.

Dr. Kinsbourne states that infantile spasms can be considered both a seizure disorder and an encephalopathy. Ex. 6, at 4. CK was started on ACTH within a week of onset. Id. at 5. Yet, instead of her outcome being optimal, she became severely developmentally delayed, with daily refractory seizures. Dr. Kinsbourne attributes CK's severe brain damage due to her continuing refractory seizure disorder. Id.

Citing articles by Jensen,[7] Baram and Haralski,[8] and Dichter,[9] Dr. Kinsbourne attributes the onset of infantile spasms to injurious or stressful stimuli which trigger the seizures in early postnatal life. Ex. 6, at 6. Relationship between encephalopathy and abnormal neuronal activity in the developing brain, by F.E. Jensen, 49 INTERNATIONAL REVIEW OF NEUROBIOLOGY 23-35 (2002) (a chapter in EPILEPSY, INFANTILE SPASMS, AND DEVELOPMENTAL ENCEPHALOPATHY (P.A. Schwartzkroin & J.M. Rho, eds. 2002). Jensen states that infantile spasms originate from a highly age-specific hyperexcitable network. Id. at 23. Jensen notes that infantile spasms most

[6] Kivity is Ex. 6-13.
[7] Jensen is Ex. 6-12.
[8] Baram and Haralski is Ex. 6-2.
[9] Dichter is Ex. 6-7.

commonly occur between the ages of three and eight months. Id. at 24. She states that glutamate is the major excitatory neurotransmitter in the brain with several subtypes of glutamate receptors. Id. at 26. In animal models, decreased expression of glutamate transporters can lead to seizures or lower seizure thresholds. Id. at 27. ɣ-Amino butyric acid ("GABA") is the predominant inhibitory neurotransmitter in the brain. Id. at 27. Jensen writes the delayed onset of functional GABAergic inhibition may contribute to the enhanced excitability of the immature brain. Id. She also states the expression of certain neuromodulatory peptides that influence neuronal excitability is developmentally regulated. Id. at 28. She thinks it possible that infantile spasms might worsen the underlying encephalopathy if one exists, leading to later neuronal injury via mechanisms such as excitotoxicity which glutamate receptors mediate. Id. at 28. Jensen notes that seizures are associated with long-term functional changes in surviving neurons, leading to a dysmature and often chronically epileptic state. Id. at 30. She suggests that maturational state, seizure activity, and subsequent encephalopathy which subtle molecular abnormalities define may interact, having a "feed forward" effect. Id. at 31.

T.Z. Baram and C.G. Haralski wrote Neuropeptide-mediated excitability: a key triggering mechanism for seizure generation in the developing brain 21 TRENDS NEUROSCI 11:471-76 (1998). Baram and Hatalski write that seizures early in life are consistent with the developing brain's excitability, and the excitatory neuropeptide corticotropin-releasing hormone ("CRH") is implicated in this triggering process. Id. at 471. They write that injurious or stressful stimuli are involved in this triggering. Id.

M.A. Dichter wrote Emerging Concepts in the Pathogenesis of Epilepsy and Epileptogenesis 66 ARCH NEUROL 4:443-47 (2009). Dichter writes that an epileptic region in the brain consists of multiple small distributed hyperexcitable networks. Id. at 444.

For a proposed model of how stress provokes infantile spasms, Dr. Kinsbourne discusses another Baram[10] article, Pathophysiology of Massive Infantile Spasms: Perspective on the Putative Role of the Brain Adrenal Axis, by T.Z. Baram, 33 ANN NEUROL 3:231-36 (1993). Baram states that Dr. West first described infantile spasms in 1841. Id. at 231. Focusing on corticotropin-releasing hormone ("CRH"), Baram posits that various types of brain injury have different effects on CRH gene expression and secretion. Id. at 233. He surmises that what in other infants would be normal stresses may result in certain infants the development of cryptogenic massive infantile spasms ("MIS") because of excessive CRH activation. Id. at 234.

For a discussion of how immune recognition of an infectious challenge rapidly activates the stress response which includes secretion of interleukin-1 ("IL-1") that activates the release of corticotropin-releasing factor ("CRF"), Dr. Kinsbourne discusses the Sapolsky[11] article, Interleukin-1 Stimulates the Secretion of Hypothalamic Corticotropin-Releasing Factor, by R. Sapolsky, et al., 238 SCIENCE (NEW SER.), 4826:522-24 (1987). Sapolsky and his co-authors state that during times of antigenic challenge to the immune system, the immune system can

[10] This Baram article is Ex. 6-1.
[11] The Sapolsky article is Ex. 6-20.

rapidly activate the stress response. Id. at 522. Chemical mediators of immunologic activation called lymphokines provoke glucocorticoid secretion. Among many lymphokines is interleukin-1 ("IL-1"), which releases substantial quantities of corticotropin-releasing factor ("CRF").

For a discussion of how vaccines engage toll-like receptors ("TLRs") to trigger an adaptive immune response, Dr. Kinsbourne discusses the van Duin article,[12] Triggering TLR signaling in vaccination, by D. van Duin, et al., 27 TRENDS IN IMMUNOLOGY 1:49-55 (2006). Van Duin and his co-authors state that TLRs are a family of pattern-recognition receptors that recognize structural components that many bacteria, viruses, and fungi share. Id. at 49.

Because the onset of CK's infantile spasms was quite abrupt, i.e., within hours, Dr. Kinsbourne assumes that TLRs were activated because only the innate immune system can mount so rapid a response. In addition, CK's neural network, being hyperexcitable, made her susceptible to these effects. Ex. 6, at 8. Dr. Kinsbourne states that once a seizure disorder begins, if it is not immediately brought under control, the child experiences "ever worsening and ultimately devastating psychomotor regression." Id. He states CK was predisposed to react adversely to stresses in infancy, which include vaccinations. Id. The purpose of vaccinations is to evoke an innate immune system response, which of necessity generates proinflammatory cytokines. Id. CK's vaccinations triggered her infantile spasms and long-lasting hyperresponsiveness of her hypothalamic-pituitary-adrenal ("HPA") axis. Id. As a result, CK has severe and mainly refractory seizures that degraded her mental development so that she is now profoundly mentally retarded. Id. at 8-9.

Dr. Kinsbourne concludes that, but for her vaccinations, CK's cryptogenic infantile spasms would not have led to such a devastating result. Id. at 9. He also writes that the one-day interval between vaccinations and first infantile spasms is medically reasonable since her innate immune system rapidly responded to the vaccinations. Id. He also states that it is plausible that routine vaccinations can trigger infantile spasms in a susceptible child, resulting in severe psychomotor regression. Id.

At the end of his opinion, Dr. Kinsbourne states that CK's vaccinations, including DTaP, significantly aggravated her pre-existing condition, the consequence being severe ongoing neurological impairments. Id. at 10.

On February 6, 2017, respondent filed the expert report of Dr. John Zempel, a pediatric neurologist and pediatric epileptologist. Ex. A. In detail, Dr. Zempel takes issue with Dr. Kinsbourne's statements. Id. Dr. Zempel's first criticism is that Dr. Kinsbourne has not shown data in medical literature that vaccinations cause infantile spasms. Id. Dr. Zempel's second criticism is that Dr. Kinsbourne's did not provide a reasonable mechanism of injury and cannot cite data in the medical literature to support his vaccine-caused mechanism of injury. Id. Dr. Zemple's third criticism is that the temporal interval between CK's vaccinations and onset of her infantile spasms is not medically reasonable because Dr. Kinsbourne did not prove that vaccines

[12] The van Duin article is Exhibit 6-26.

are the cause. Since children receive vaccines at the time they develop infantile spasms, some spasms will necessarily occur in close temporal relationships to vaccination, which is why epidemiologic studies are necessary to evaluate causation. Id. Dr. Zempel's fourth criticism is that the absence of an alternative cause of CK's infantile spasms does not prove vaccine causation. Many children have intractable infantile spasms of unknown cause. Id. Dr. Zempel's fifth criticism is that Dr. Kinsbourne has not specifically shown biologic plausibility through medical literature describing experiments on animal models to prove vaccinations cause infantile spasms in humans, or through data from human diagnostic testing. Id. Dr. Zempel continues by emphasizing it is important to show mechanistically or in medical literature such as or epidemiologic studies that vaccination causes infantile spasms. Id.

Dr. Zempel's approach is to analyze Dr. Kinsbourne's statements to see if medical literature supports them. Id. at 6. Dr. Zempel does not accept any statement that does not have support in the medical literature. Id. Dr. Zempel disagrees with Dr. Kinsbourne's relying on studies such as the NCES from the 1970s that discuss whole-cell DTP vaccine and infantile spasms because they do not discuss acellular DTP, i.e., DTaP. Id. Dr. Zempel quotes the concluding paragraph of the 1983 Bellman study (Tab 1), based on the NCES data, which concludes that pertussis vaccine is not a direct cause of infantile spasms but may precipitate the onset of spasms in those children already destined to develop them. Id.

Dr. Zempel then quotes the conclusion of the abstract from the 1977 Melchior study (Tab 2) analyzing the difference in onset of infantile spasms after Denmark changed the vaccine schedule for infants. Id. at 7. Melchior wrote there was no change in age of onset of infantile spasms, but admitted there may be an occasional connection between immunization and infantile spasms, which he attributed to coincidence. Id.

Dr. Zempel then quotes a chapter from Aicardi in a textbook in epilepsy in children by Arzimangoglou (Tab 3) to the effect that onset of infantile spasms after immunization is coincidental. Id. Dr. Zempel says that medical literature does not support Dr. Kinsbourne's opinion that CK would not have developed infantile spasms without her having been vaccinated. Id. If she had, since they were cryptogenic, her outcome would have been better, which Dr. Zempel states is assuming causation in the first place. Id. at 7-8.

Dr. Zempel agrees that children with cryptogenic infantile spasms have better outcomes than children with symptomatic infantile spasms. Id. at 8. Dr. Kinsbourne writes that CK's profound impairment puts her at the severe end of the spectrum of infantile spasms outcome, which is quite atypical for cryptogenic infantile spasms. Id. Dr. Zempel does not believe CK would have ever been normal developmentally. Id. at 9. In his clinical experience, Dr. Zempel has treated many cryptogenic infantile spasms patients who do not have a good outcome developmentally although they received similar treatment as CK, except for cannabidiol. Id. He cites two articles, both of which have the primary author as Knupp,[13] for the proposition that a

[13] Response to second treatment after initial failed treatment in a multicenter prospective infantile spasms cohort, by K.G. Krupp, et al., 57 EPILEPSIA 11:1834-42 (2016) (Tab 8); Response to treatment in a prospective national

sizable number of children with infantile spasms are unresponsive to drugs. Id.

Dr. Zempel cites data demonstrating that good neurodevelopmental outcome is not dependent on successful treatment of infantile spasms, whether or not they are cryptogenic. Id. at 10. He criticizes Dr. Kinsbourne for opining that vaccination caused CK's infantile spasms or worsened their outcome when Dr. Kinsbourne did not provide epidemiologic support or strong mechanistic data. Id. at 12. Dr. Zempel states that such evidence is not present in medical literature. Id.

Dr. Zempel notes that because infantile spasms occur in a particular age window, this strongly suggests that developmental gene expression shapes their appearance during a specific time in human development. Id. at 12-13. Specific mechanisms responsible for the development of seizures in general are still the subject of current epilepsy research. Id. at 13. Animal models of infantile spasms are an aspirational goal. Id. Dr. Zempel then analyzes Dr. Kinsbourne's "two-hit" theory, whereby CK was born with a susceptibility to develop infantile spasms (the first hit) and the vaccinations (the second hit) triggered their onset. Dr. Zempel rejects the first hit, i.e., CK's susceptibility, because basic research has not determined the mechanism for infantile spasms. Dr. Zempel rejects the second hit, i.e., the role of vaccinations in causing or triggering infantile spasms, because Dr. Kinsbourne does not cite objective independent evidence from treating physicians or the medical literature. Although Dr. Zempel accepts that vaccination provokes an immune response, he does not accept that this provoked immune response is a risk factor for causing infantile spasms. Id.

Dr. Zempel states that CK clearly has drug-resistant infantile spasms which are closely associated with developmental delay. Id. at 17. Dr. Zempel also states he does not know the etiology of her infantile spasms. Id. Dr. Zempel agrees that CK's neurodevelopmental issues are clearly related to her intractable and drug-resistant infantile spasms. Id. at 20.

On April 10, 2017, petitioners filed Dr. Kinsbourne's supplemental expert report. Ex. 6-A. He admits that scientific proof is lacking that vaccinations can cause infantile spasms, but he says his opinion is based on a reasonable degree of medical probability. Id. at 1. Referring to Dr. Zempel's quotations from the Bellman study and the Melchior study, Dr. Kinsbourne states that saying vaccines may precipitate infantile spasms is the same as saying vaccines may cause infantile spasms. Id. Melchior even says there may be an occasional connection between vaccines and infantile spasms. Id. Dr. Kinsbourne notes that no explanatory model for the genesis of infantile spasms has been scientifically proven. Id. at 3.

## TESTIMONY

CK's mother testified first. Tr, at 4. She used to be an emergency department nurse. Id. at 5. She and her husband have five children. Id. at 6. CK seemed normal for four months. Id. at 8. CK's mother brought CK to the pediatrician in the morning for her four-month well-baby

infantile spasms cohort, by K.G. Krupp, et al., 79 ANN NEUROL 3:475-84 (2016) (Tab 9).

checkup where CK received her vaccinations. Id. That evening, while CK's mother was nursing CK, CK's arms and head went forward and her legs came up. This was her first cluster of seizures. Id. CK's father called the pediatrician who told him to bring CK into the emergency room right away. Id. CK has never run a temperature with her seizures. Id. at 13. Dr. Riordan, CK's pediatrician, thought CK had either gastroesophageal reflux or a reaction to her vaccinations. Id.

CK's mother said that she and her husband have watched CK deteriorate with every seizure. Id. at 14. CK withdrew eye contact and has a hard time interacting with the world. She does not speak. She walks with severe ataxia and will walk into objects and seize into the ground. CK's parents have watched CK go from a normal, healthy, developing child to a catastrophically ill one. Id. Nothing has eradicated CK's seizures. Id. at 15. CK currently seizes from 30 to 50 times a day. Id.

The next witness was petitioners' expert, Dr. Marcel Kinsbourne, a pediatric neurologist. Id. at 32, 35. He has seen hundreds of cases of seizure disorder in his professional career. Id. at 36. He has seen 20 to 40 infantile spasms cases in his professional career. Id. He was a resident at the Great Ormond Street Hospital for Sick Children [located in London, England] when the first studies were done on whether ACTH benefits children with infantile spasms. Id.

Dr. Kinsbourne's opinion is that CK's vaccinations caused or triggered her infantile spasms that occurred 10 hours later. Id. at 38. His basis is that CK was a normal baby before the vaccinations and, after vaccinations, she had a very definite, clear onset of a cluster of 30 or 40 successive spasms within hours. Id. at 39. In addition, vaccines discharge proinflammatory cytokines to stimulate the innate immune system to produce a response but in CK, this response was infantile spasms. Id. at 41. Pertussis vaccine is known to be epileptogenic at times. Id. at 43. Although in acellular DPT, the pertussis is toxoided, there is still some high-toxoided toxin in it. Id. Dr. Kinsbourne accepts that although DTaP is less reactogenic than whole-cell DTP, recipients of DTaP can still have adverse reactions. Id.

Dr. Kinsbourne said 10 hours between CK's vaccinations and her onset of infantile spasms was very appropriate for causation because innate immune system reactions are very fast. Id. at 44. He also said there was a logical sequence of cause and effect between CK's receipt of four vaccines, including DTaP, well-known to be capable of stimulating the innate immune system, and the production of proinflammatory cytokines which can occur even without causing a fever. Id. at 44-45.

Dr. Kinsbourne was impressed with the clear and decisive manner of the onset of CK's infantile spasms as infantile spasms do not usually begin that way. Id. at 46. Infantile spasms often have an insidious onset. Id. at 70. CK's abrupt onset of infantile spasms suggests to Dr. Kinsbourne that a definite event provoked the seizures. Id. at 47.

Dr. Kinsbourne testified that infantile spasms destroy the brain. Id. at 48. CK is very

typical as a demonstration of that because infantile spasms did not immediately affect her development. Id. at 49. As CK's seizures got worse and her medicines became less effective, her development became worse and worse. Id. at 49. Her seizures still continue and, thus, CK never had the chance to develop as a normal child. Id. Dr. Kinsbourne said if CK had not received her vaccinations at the age of four months but when she was substantially older and then began to seize, her brain damage would not be as severe as it is now. Id. Later in life, CK's seizures would have taken a form that is not as damaging to the brain as infantile spasms. Id. at 59.

Dr. Kinsbourne said that CK's infantile spasms are cryptogenic because no one knows the cause of them. Id. If we knew the cause of them, she would have symptomatic infantile spasms, which are more common. Id. at 49-50.

Dr. Kinsbourne described his "two-hit" theory. Id. at 51. The first hit is that something is different in CK's brain to make her susceptible to vaccination. Id. The second hit is the risk factor, in this case, the vaccinations, which triggered the onset of the infantile spasms. Id. He posited that if CK had not received these vaccines, either she would not have had infantile spasms or she would have had them at a later age and had a better outlook for development. Id. at 53. The amount of brain damage depends on how early someone starts to have infantile spasms. Id. Dr. Kinsbourne saw CK seize the day before the trial and testified she still has severe seizures. Id. at 54. They are still infantile spasms. Id.

The undersigned asked Dr. Kinsbourne if he agreed that the results of the Bellman, Ross, and Miller study which compared the incidence of cryptogenic infantile spasms among children who received whole-cell DPT with the baseline occurrence of children the same age who had cryptogenic infantile spasms and concluded that DPT triggered infantile spasms among DPT vaccinees within the first week of vaccination was applicable to children who receive acellular DPT, just at a lower incidence. Id. at 55-57. Dr. Kinsbourne said he absolutely agreed. Id. at 57. He also agreed that the conclusion of the Bellman study that DPT vaccination triggers onset of cryptogenic infantile spasms if they occur within one week of vaccination applied to CK because her onset was within one week of vaccination. Id. He said that the Bellman study shows clearly that pertussis vaccine can trigger the onset of infantile spasms. Id. at 58.

The third witness was respondent's expert Dr. John Zempel, a pediatric neurologist and pediatric epileptologist. Id. at 106. He has seen thousands of patients with epilepsy over the last 15 years with 10 to 15 of them per year having infantile spasms. Id. at 110, 113. He defined infantile spasms as an age-dependent epileptic encephalopathy. Id. at 111. An overall encephalopathy coexists with the seizures. Id. Parents are very concerned about their children having infantile spasms because of the dire developmental outcomes occurring in these patients. Id. at 112. A small fraction of children with infantile spasms respond to aggressive treatment. Id. at 113. CK's case is unusual because she has refractory or drug-resistant infantile spasms and did not have a remission. Id. at 114.

Dr. Zempel's opinion is that CK's vaccinations did not cause or exacerbate her infantile spasms. Id. at 118. The basis of his opinion is his clinical experience and the medical literature in which doctors broadly do not recognize vaccination as a cause of infantile spasms. Id. Dr. Zempel said that Bellman's study does recognize a causal factor, but it is an old paper. Dr. Zempel stated the more recent medical literature does not include vaccination as a likely or even uncommon cause of infantile spasms. Id. He said the preferred current way of viewing cryptogenic infantile spasms is that they have no identified etiology, i.e., there must be an etiology but it is as yet unknown. Id. at 119. In other words, there is an underlying propensity in the population of these children to have infantile spasms. Id.

Dr. Zempel said that infantile spasms have a unique identity and mostly tend to cluster when the children are waking or falling asleep. Id. at 120-21. Each seizure lasts a few seconds and may recur every 30 seconds, every minute, or every several minutes for a period of time. Id. at 121. Because of the dire outcome associated with infantile spasms, pediatric neurologists are well-trained to recognize infantile spasms. Id. An EEG particularly during the sleep phase will capture the high-amplitude chaos in the brain known as hypsarrhythmia. Id. at 123. This means the entire brain is not working well which is what encephalopathy means. Id. Because doctors want to diagnose infantile spasms expeditiously in order to get the child into treatment, infantile spasms are the only seizure disorder for which a hospital will do an EEG at night or on a Saturday or Sunday for diagnostic purposes. Id. Generally, infantile spasms start between three to nine months of age. Id.

Dr. Zempel thinks that both the infantile spasms and the encephalopathy destroy the brain. Id. at 127. The medical community thinks that more severe infantile cases have earlier onset. Id. at 131. In most people with epilepsy, we do not know why they have a seizure each time they seize. Id. at 137. Stress can cause a seizure but we do not know how. Id. at 138.

Dr. Zempel said we do not know the mechanism by which any epilepsy occurs. Id. at 144. A seizure occurs as a hyperexcitability of neuronal circuits but we do not know why. Id. Dr. Zempel said circuits are a group or a network of neurons that are discharging, like an electrical storm. Id. But then during this electrical storm, there is a breakdown of normal mechanisms that keep that electrical activity in a small area. Id. at 144-45. How big the seizure becomes or where it spreads defines many types of epilepsies. Id. at 145. In a classic case of hypsarrhythmia, there is chaos everywhere in the brain. Id. In some cases of infantile spasms, there is focality, but in other cases, there is not. Id. at 146. Those who read CK's EEGs found more focality at various times and then less focality at other times. Id.

Dr. Zempel discussed the Coppola article,[14] not mentioned in his expert report, but filed as Exhibit E, which describes three sets of identical twins whose onset of infantile spasms was essentially on the same day of each twin's life, a strong argument for a genetic determinant. Id. at 150-51. He thinks that genetics influences all infantile spasms. Id. at 153. Some are likely

[14] Simultaneous Onset of Infantile Spasms in Monozygotic Twins, by G. Coppola, et al. 43 PEDIATR NEUROL 127-30 (2010). Ex. E. The long-term outcome was poor in all six twins. Id. at 130.

directly causative. Id. Other cases may have indirect genetic causation. Id. at 154. He believes CK has some underlying condition of her brain. Id. at 182.

Dr. Zempel stated that vaccinations are not known in the medical community to be a cause of infantile spasms. Id. at 160. He thinks that recall bias explains the temporal shift in onset of infantile spasms after DPT which the Bellman study reflects. Id. at 161. Dr. Zempel said the statistics in the Bellman paper are not very statistically significant. Id. at 162. It depends on a very small number of cases. Id. As to whether or not CK's outcome would have been better if her onset of infantile spasms occurred later than it did, Dr. Zempel replied that we do not truly know on a day-to-day, week-to-week, or perhaps even month-to-month basis that timing influences ultimate outcome. Id. at 163-64. "It is always better to get rid of seizures earlier." Id. at 164. But Dr. Zempel questioned how important it is to get rid of infantile seizures as rapidly as possible because of the variety of treatments for them which involve different improvement times, if they work at all. Id. at 163, 164. He said there is a dearth of long-term data in studies looking at longer term outcomes. Id. at 164.

Dr. Zempel testified that there is a small fraction of children with cryptogenic infantile spasms that do better than symptomatic infantile spasms children, but not a majority of them. Id. at 165. The evolving opinion of the medical community is that someone with infantile spasms has an abnormal brain. Id. Using the diagnosis of cryptogenic infantile spasms means doctors just do not understand yet why that particular child has infantile spasms. Id. Dr. Zempel said that one of the depressing thoughts is that things are not that much better now than they were in the prior era for children with infantile spasms. Id. at 168. He thinks that CK had a very typical presentation and initial course of infantile spasms. Id. at 169. But by being drug-resistant and continuing to have spasms beyond a typical age when they remit, CK is unfortunately in a more selective category of infantile spasms that have not remitted. Id. The fact that she is having brief seizures has significant consequences. Id.

Dr. Zempel said that steroids, like ACTH, do not just modulate the immune system; they directly modulate the brain when used in infantile spasms cases. Id. at 171. He thinks whether the immune system has a pathogenic role in the development of infantile spasms is a complicated question. Id. Dr. Zempel thinks CK got excellent treatment with aggressive identification of her illness and institution of ACTH. Id. at 172.

Dr. Zempel said that medical literature in the form of peer-reviewed articles does not exist for vaccine causation of infantile spasms. Id. at 176. One of the holy grails, as Dr. Zempel termed it, of pediatric epilepsy research is to develop an animal model of infantile spasms, particularly rodent models. Id. at 177. He said it is very, very difficult to created animal models for infantile spasms because the rat or mouse brain may be different than the human brain. Id. The only experiments used on animals to try to develop something that looks like infantile spasms has been by doctors using neurotoxins as very severe insults to the brains of young animals. Id. at 178-79. But whether these animal experiments truly reflect infantile spasms is a very complicated and very controversial subject. Id. at 179. The new term for infantile spasms

is epileptic spasms. Id. at 180.

## DISCUSSION

To satisfy their burden of proving causation in fact, petitioners must prove by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen v. Sec'y of HHS, 418 F.3d 1274, 1278 (Fed. Cir. 2005). In Althen, the Federal Circuit quoted its opinion in Grant v. Secretary of Health and Human Services, 956 F.2d 1144, 1148 (Fed. Cir. 1992):

> A persuasive medical theory is demonstrated by "proof of a logical sequence of cause of and effect showing that the vaccination was the reason for the injury [,]" the logical sequence being supported by a "reputable medical or scientific explanation[,]" i.e., "evidence in the form of scientific studies or expert medical testimony[.]"

418 F.3d at 1278.

Without more, "evidence showing an absence of other causes does not meet petitioner's affirmative duty to show actual or legal causation." Grant, 956 F.2d at 1149. Mere temporal association is not sufficient to prove causation in fact. Id. at 1148.

Petitioners must show not only that but for CK's vaccinations, she would not have infantile spasms and chronic neuropathy, but also that her vaccinations were substantial factors in causing her infantile spasms and chronic neuropathy. Shyface v. Sec'y of HHS, 165 F.3d 1344, 1352 (Fed. Cir. 1999).

In Capizzano v. Sec'y of HHS, 440 F.3d 1317, 1325 (Fed. Cir. 2006), the Federal Circuit said: "we conclude that requiring either epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect is contrary to what we said in Althen . . . ." Such an approach is inconsistent with the use of circumstantial evidence. Id. The Federal Circuit in Althen rejected the assertion that the Vaccine Act;s preponderant evidence standard requires objective confirmation. 418 F.3d at 1279. The Federal Circuit stated that "the purpose of the Vaccine Act's preponderance standard is to allow the finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body." Id. at 1280.

Close calls are to be resolved in favor of petitioners. Capizzano, 1440 F.3d at 1327; Althen, 418 F.3d at 1280. In Althen, the Federal Circuit ruled in favor of a causal link between tetanus toxoid vaccine and optic neuritis and acute disseminated encephalomyelitis, which it recognized was "a sequence hitherto unproven in medicine." Id.

In essence, the special master is looking for a medical explanation of a logical sequence of cause and effect (Althen, 418 F.3d at 1278; Grant, 956 F.2d at 1148), and medical probability rather than certainty (Knudsen v. Sec'y of HHS, 35 F.3d 543, 548-49 (Fed. Cir. 1994)). To the undersigned, medical probability means biologic credibility rather than specification of an exact biologic mechanism. As the Federal Circuit stated in Knudsen:

> Furthermore, to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program. The Vaccine Act does not contemplate full blown tort litigation in the Court of Federal Claims. The Vaccine Act established a federal "compensation program" under which awards are to be "made to vaccine-injured persons quickly, easily, and with certainty and generosity." House Report 99-908, [99th Cong. 2d Sess. 18], at 3, 1986 U.S.C.C.A.N. at 6344.
>
> The Court of Federal Claims is therefore not to be seen as a vehicle for ascertaining precisely how and why DTP and other vaccines sometimes destroy the health and lives of certain children while safely immunizing most others.

35 F.3d at 549.

As for epidemiological support for causation, the Federal Circuit in Knudsen, 35 F.3d at 551, ruled for petitioners even when epidemiological evidence directly opposed causation from DPT vaccine. The case concerned the cause of a baby's encephalopathy after a vaccination. Respondent provided evidence that more encephalopathies are caused by viruses than by vaccines, convincing the special master to rule against petitioners. But the Federal Circuit thought the epidemiologic evidence should not bar petitioners from prevailing. Even though epidemiological evidence supported respondent's view that viruses were more likely to cause encephalopathy than vaccinations, the Federal Circuit held that that fact alone was not an impediment to recovery of damages. In Knudsen, the Federal Circuit stated:

> The bare statistical fact that there are more reported cases of viral encephalopathies than there are reported cases of DTP encephalopathies is not evidence that in a particular case an encephalopathy following a DTP vaccination was in fact caused by a viral infection present in the child and not caused by the DTP vaccine.

35 F.3d at 550.

Interestingly, the Federal Circuit in Knudsen also stated that when a vaccinee would fit within an epidemiological study, that alone is sufficient proof of vaccine causation:

> Causation in fact under the Vaccine Act is thus based on the circumstances of the particular case, having no hard and fast *per se* scientific or medical rules. The determination of causation in fact under the Vaccine Act involves ascertaining whether a sequence of cause and effect is "logical" and legally probable, not medically or scientifically certain. [citing cases] Thus, for example, causation can be found in vaccine cases based on epidemiological evidence and the clinical picture regarding the particular child without detailed medical and scientific exposition on the biological mechanisms. [citing case].

Id. at 548, 549.

Both Dr. Kinsbourne for petitioners and Dr. Zempel for respondent agreed on a number of issues. CK's cryptogenic infantile spasms have an unknown cause. In addition, before she received her four-month vaccinations, her brain was abnormal even though her behavior until her four-month vaccinations was normal. For reasons scientists and doctors have not been able to discover, within hours of her four-month vaccinations, CK had her first infantile spasms cluster. She has been seizing ever since, even though the expectation is that aggressive treatment, such as ACTH, will stop the spasms, or even getting older than eight or nine months will lead to normalcy. CK kept seizing. She is now severely delayed in all categories and she still has infantile spasms.

Respondent defends based on all the criteria that the Federal Circuit has rejected in Knudsen, Althen, and Capizzano: (1) we do not know the specific mechanism for how vaccines can cause cryptogenic infantile spasms or the biological mechanisms explaining why cryptogenic infantile spasms occur at all; (2) we do not have epidemiological studies that confirm that vaccines can cause cryptogenic infantile spasms; (3) we do not have animal models of vaccines causing cryptogenic infantile spasms; (4) whether CK seized early in life (at four months) or later makes no difference because we cannot predict the outcome of a child who has cryptogenic infantile spasms; (5) the Bellman study (1983) used a very small number of cryptogenic infantile spasms children to determine that whole-cell DPT triggered the vaccinees's onset of infantile spasms within one week of vaccination; and (6) the Melchior study (1977) found just a few infantile spasms related to DPT vaccination, below statistical significance. The undersigned rejects all of these criteria as not being consistent with the Federal Circuit's repeated guidance in causation in fact cases.

Both parties filed the Bellman study (1983) and the Melchior study (1977). Pet'rs' Ex. 6-4; Resp't's Ex. D, Tab 1, and Pet'rs' Ex. 6-16; Resp's Ex. D, Tab 2. The undersigned asked petitioners' expert Dr. Kinsbourne if adverse reactions to acellular DPT (which CK received) could occur, but just at a lower incidence than adverse reactions to whole-cell DPT. He said yes. The undersigned finds Dr. Kinsbourne's opinion credible and finds that CK would have fit into the Bellman study which, based on a week-by-week analysis, found that among cryptogenic

infantile spasms vaccinees, their onset of infantile spasms occurring within the first week after vaccination was higher than baseline cryptogenic infantile spasms children. Because the onset of cryptogenic infantile spasms vaccinees within the second week after vaccination was lower than baseline cryptogenic infantile spasms children, Bellman concluded that DPT vaccine was a trigger to the onset of infantile spasms in children so that the spasms occurred sooner than they would have without vaccination, but also that the children were destined to have infantile spasms. Bellman concluded that pertussis immunization "is not a direct causal factor for infantile spasms in children with structurally normal brains, but . . . it may precipitate the onset of spasms in those children in whom the disorder is already destined to develop." Pet'rs' Ex. 6-4, at 1033; Resp't's Ex. D, Tab 1, at 1033.

Similarly in the Melchior study (1977), Melchior analyzed the number of infantile spasms after Denmark changed its vaccination schedule and concluded that there were three cases of infantile spasms in which vaccination could be considered as a triggering mechanism. Pet'rs' Ex. 6-16, at 135; Resp't's Ex. D, Tab 2, at 135. Neither Bellman nor Melchior viewed their respective triggering conclusions as based on statistical significance. Melchior concluded that a causal connection between DPT and infantile spasms was very unlikely "except in a few cases." Id. at 136.

Does it make any difference that DTaP was a trigger rather than a cause of CK's infantile spasms? The undersigned asked petitioner's expert Dr. Kinsbourne if CK's having infantile spasms at the age of four months was worse for her ultimate outcome than if she had had them when she was older, i.e., if she was, as Bellman concluded, destined to have infantile spasms anyway? He answered in the affirmative. The seizures destroy the brain. The concern, which Dr. Zempel shared, with children with infantile spasms is to treat them aggressively because infantile spasms do tremendous damage to the brain. The evidence of the horrible outcome of these seizures is that CK is irreparably damaged. That doctors may not know looking forward what the outcome of an infantile spasms child would be is irrelevant when we already know the outcome of CK's infantile spasms.

Putting this all together, the undersigned finds that CK, even though she received DTaP, not DPT, would have qualified to have been in the Bellman and Melchior studies because she had infantile spasms within a week of pertussis vaccination and the vaccination was a trigger, according to both the Bellman and Melchior studies, which prompted the onset of her spasms. We are not dealing with the niceties of statistical significance in the Vaccine Program under the guidance of the Federal Circuit's decisions in Knudsen, Althen, and Capizzano. The principle the Federal Circuit pronounced in Knudsen, i.e., that causation can be found in vaccine cases based on epidemiological evidence and the clinical picture regarding the particular child without detailed medical and scientific exposition on the biological mechanisms governs the outcome of this decision.

Because CK would have qualified to have been in the Bellman and Melchior studies, the undersigned finds that her four-month vaccinations triggered the onset of her cryptogenic

seizures. The undersigned further finds that but for her onset of cryptogenic infantile spasms at four months of age, she would have not had the disastrous outcome she had.

Similar analysis is in H.J. v. Sec'y of HHS, No. 110301V, 2015 WL 6848357 (Fed. Cl. Spec. Mstr. Nov. 6, 2015), in which petitioner prevailed on the theory that Tdap (tetanus-diphtheria-acellular pertussis) vaccine triggered her rheumatoid arthritis ("RA") based on the theory that an environmental trigger such as a vaccine can cause preclinical RA to develop into clinical RA. 2015 WL 6848357, at *10. In the instant action, CK was clinically normal, but both Dr. Kinsbourne and Dr. Zempel agreed that CK had an abnormal brain. Similarly, in H.J., petitioner had pre-vaccination autoimmune diseases, but not RA. H.J.'s expert stated that further proof of vaccine causation was that H.J.'s onset of RA was explosive, unlike the usually insidious onset of RA. Id. at *11. The special master in H.J. accepted petitioner's expert's explanation for H.J.'s abrupt onset of RA. Id. at 12.

Similarly, Dr. Kinsbourne in the instant action focused on the abrupt onset of CK's cryptogenic infantile spasms as further proof of DTaP vaccine causation. As both he and Dr. Zempel testified, the usual onset of infantile spasms is insidious. But CK's onset of cryptogenic infantile spasms was explosive, sudden, and dramatic. This proved to Dr. Kinsbourne that DTaP was a trigger of CK's cryptogenic infantile spasms. The undersigned finds Dr. Kinsbourne's testimony credible and accepts his opinion on causation in CK's case as further support for the Bellman study and Melchior study conclusions that pertussis vaccine can trigger the onset of infantile spasms.

The undersigned need not comment on Dr. Kinsbourne's two-hit theory in that both experts agree that CK brain was abnormal pre-vaccination and the undersigned finds that DTaP triggered the onset of CK's cryptogenic infantile spasms. Moreover, as Dr. Zempel testified, CK has a chronic encephalopathy.

The undersigned is satisfied with the evidence in the record that the medical literature acceptance of pertussis vaccine as a trigger to onset of infantile spasms in a few cases within one week of vaccination is sufficient to prove causation in this case, buttressed by the evidence of an explosive onset of infantile spasms rather than the normal insidious onset of infantile spasms.

Althen Analysis

Under Althen Prong One, the undersigned finds that DTaP vaccine can trigger the onset of infantile spasms. Under Althen Prong Two, the undersigned finds that there was a logical sequence of cause and effect in DTaP causing CK's onset of infantile spasms. Under Althen Prong Three, the undersigned finds that an onset within hours of DTaP vaccination is consistent with the effect of the vaccine's triggering an abrupt onset.

The undersigned rules in favor of petitioners on entitlement. This case is now in damages. The undersigned will schedule a status conference soon to discuss how the parties will

proceed with damages. Because of their prior life care plans, there should be an expedited process to settle damages. Since the prior settlement negotiations that failed, petitioners have bought a house, raising the issue of whether or not there needs to be any house modification. The undersigned orders petitioners to file updated medical records and IEPs, as well as any other information relevant to the issue of damages, including the existence of any Massachusetts Medicaid lien.

**IT IS SO ORDERED.**

Dated: December 12, 2017

/s/ Laura D. Millman
Laura D. Millman
Special Master